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Spontaneous recovery of consummatory behavior, but not of consummatory successive negative contrast ☆

Jacob N. Norris, Alan M. Daniel, Mauricio R. Papini *

Department of Psychology, Texas Christian University, P.O. Box 298920, Fort Worth, TX 76129, USA

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ABSTRACT

Five experiments were designed to study spontaneous recovery (SR) in two situations involving consummatory behavior: consummatory successive negative contrast (cSNC) and consummatory extinction (cE). SR of consummatory suppression should occur if incentive downshift induces an egocentric memory encoding information about the emotional reaction to the downshift that is counterconditioned or extinguished during exposure to the downshifted reward. SR of cSNC failed to occur after resting periods of 24, 96, or 336 h interpolated following complete (Experiment 1) and incomplete (Experiment 2) recuperation of consummatory behavior, and was not induced by the opioid-receptor antagonist naloxone (2 mg/kg), known to enhance cSNC (Experiment 3). However, SR of consummatory behavior occurred across sessions in cSNC (Experiment 3) and cE (Experiments 4–5). Furthermore, naloxone facilitated cE without affecting SR (Experiments 4–5). These results are discussed in relation to evidence for the development of an egocentric memory of the aversive downshift experience in consummatory situations.

Surprising downshifts in incentive quality or quantity often lead to aversive emotional arousal. Traditionally, the emotion triggered by unexpected reductions of appetitive incentives has been labeled frustration (Amsel, 1992). Surprising reward reductions have been extensively studied in the context of two basic procedures differentiated by the extent of the loss (Mackintosh, 1974). In successive negative contrast (SNC) the loss is partial as the incentive is reduced to a nonzero value, whereas in appe-

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* Corresponding author. Fax: +1 817 257 7681.
E-mail address: m.papini@tcu.edu (M.R. Papini).

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titive extinction the loss is complete as the incentive is withheld. The present experiments involve consummatory versions of these two procedures. In consummatory SNC (cSNC), rats receive access to a 32% sucrose solution for a number of sessions (typically 10 daily sessions, 5-min each) and then the concentration is downshifted to 4% sucrose for an additional 4–5 postshift sessions. The consummatory performance of downshifted rats during these postshift sessions is significantly lower than that of an unshifted control group exposed always to 4% sucrose during the entire experiment (Flaherty, 1996; Vogel, Mikulka, & Spear, 1968). In consummatory extinction (cE), sessions of access to sucrose solution (usually 32% or 4% sucrose) are followed by sessions of access to an empty tube. Consummatory behavior decreases both between and within sessions (Mustaca, Freidin, & Papini, 2002).

Adjustments to situations involving incentive downshifts might depend on at least two types of memory called allocentric and egocentric (Papini, 2003). *Allocentric memories* are established when the organism tracks changes in the environment and updates the memory record of the situation. For example, animals exposed to an incentive downshift eventually respond to a level appropriate to the new magnitude as they update their memory record of the situation to reflect the absence of reinforcement in a situation that used to produce it. *Egocentric memories* are established when the organism learns to anticipate its own emotional reaction to the incentive change. Using again the example of the incentive downshift, egocentric learning can modulate the speed of behavioral change causing a drastic deterioration in anticipatory behavior, as in instrumental SNC (iSNC). In the traditional terms of learning theory, allocentric memory relates to expectancies of reinforcement, whereas egocentric memory relates to such internal emotional responses as fear and secondary frustration (Amsel, 1992; Mowrer, 1960).

The present experiments center on the issue of whether an aversive egocentric memory is acquired during the initial downshift experience, characterized behaviorally by consummatory suppression. The alternative hypothesis maintains that cSNC and cE can be explained entirely on the basis of an unconditioned reaction to reward loss, akin to Amsel, 1992 primary frustration, combined with an allocentric memory of the preshift incentive. According to this view, neither cSNC nor cE would involve the development of an aversive egocentric memory, but just a session-to-session induction of primary frustration. Recuperation[1] from cSNC and behavioral extinction during cE reflect a reduction of primary frustration resulting from the allocentric update of incentive value. Such update would lead to a progressive decrease in the strength of primary frustration as the current value becomes better predicted by the reactivation of the updated allocentric memory.

Potential (but inconclusive) evidence for egocentric memory in situations involving unexpected incentive reductions comes entirely from cSNC experiments. First, pharmacological evidence suggests that at least two types of drugs, benzodiazepine anxiolytics and opioids, can have behaviorally selective effects on cSNC. For example, benzodiazepine anxiolytics including chlordiazepoxide, flurazepam, diazepam, and midazolam, have been shown to reduce to cSNC only when administered after the organism has accumulated some experience with the downshifted solution (Becker, 1986; Becker & Flaherty, 1983; Flaherty, Becker, Checke, Rowan, & Grigson, 1992; Mustaca, Bentosela, & Papini, 2000). Similar behavioral selectivity occurs with opioid treatments. For example, the δ-opioid-receptor agonist D-Ala2-N-MePhe4,Gly-ol (DPDPE) reduces cSNC during the first postshift session (when there is minimal experience with the downshifted solution), but not during the second postshift session (Wood, Daniel, & Papini, 2005), whereas naltrindole, a δ-opioid-receptor antagonist, enhances cSNC when administered before the first postshift session, but has no effect on the second postshift session (Pellegrini, Wood, Daniel, & Papini, 2005). Conversely, the κ-opioid-receptor agonist U50,488H reduces contrast when administered on the second postshift trial, but is ineffective on the first postshift trial (Wood, Norris, Daniel, & Papini, in press). Session selectivity suggests the possibility that these drug effects depend on the acquisition of an egocentric memory of the downshifted incentive, but do not prove it. For example, the benzodiazepine chlordiazepoxide has no effect on cSNC when administered before the first downshift session, except when the session is extended from the

[1] To avoid confusion with "spontaneous recovery," "recuperation" is used instead of the more common "recovery" to describe the behavioral changes that occur during the postshift sessions, after the incentive downshift.

typical 5–20 min of duration (Flaherty, Grigson, & Rowan, 1986) or after repeated downshifts (Flaherty, Clark, & Coppotelli, 1996). While these examples of session specificity of drug effects are consistent with the hypothesis of an egocentric aversive memory, they do not provide unambiguous evidence because more than one change in memory is occurring during these postshift sessions. In addition to the aversive experience of the loss, animals are presumably updating their record of the situation. Thus, trial specificity of a drug may reflect an effect on the allocentric updating process, rather than on the egocentric encoding of the loss experience.

A second source of potential evidence for egocentric memory comes from experiments involving posttrial drug administration. This procedure has been extensively used to identify neurochemical systems involved in memory consolidation. For example, the role of the cholinergic system on memory consolidation in a variety of tasks, including iSNC, was identified using postsession drug administration (e.g., Salinas, Introini-Collison, Dalmaz, & McGaugh, 1997). When administered immediately after the first postshift session, cholinergic drugs failed to affect the subsequent course of the cSNC effect (Bentosela et al., 2005). Similarly, postsession 11 administrations of naloxone, naltrindole, and DPDPE fail to influence cSNC (Daniel, Ortega, & Papini, in preparation). A similar experiment with U-50,488H (3 mg/kg, ip) did lead to an enhancement of cSNC, but additional evidence suggested that this effect was probably due to the induction of an aversion to the 4% sucrose by the contiguous pairing with U-50,488H (Wood et al., in press). So far, the only evidence of postsession modulation of cSNC comes from corticosterone administration (Bentosela, Ruetti, Muzio, Mustaca, & Papini, 2006). This effect occurs when corticosterone is administered immediately at the end of session 11, but not when it is administered 3 h later. Additional data confirm that corticosterone does not support conditioned taste aversion when administered under the same conditions of training, but in the absence of an incentive downshift (Ruetti, Justel, Mustaca, & Papini, submitted for publication). Exactly how corticosterone achieves this effect on cSNC remains unclear, although one possibility is that it facilitates the consolidation of an egocentric memory encoding aversive information about the downshift experience. The enhancing effects of adrenal stress hormones on the consolidation of aversive memories is well documented (McGaugh, 2000). However, postsession 11 corticosterone may be enhancing cSNC through mechanisms unrelated to the aversive egocentric memory of the downshift, including a potential facilitatory effect on search behavior that may prolong the cSNC effect (Pecoraro, Gomez, & Dallman, 2005).

The present experiments represent a third approach to determining whether the incentive downshift manipulation involves the acquisition of an aversive egocentric memory. Acquired behaviors generally show one major property: spontaneous recovery. Spontaneous recovery (SR) occurs when an extinguished or a counterconditioned response to a conditioned stimulus restores part of its former strength after a resting period without further stimulation. SR of conditioned responses occurs in a variety of Pavlovian conditioning situations, whether after extinction (Pavlov, 1927), aversive-to-appetitive counterconditioning (Bouton & Peck, 1992), or partial reinforcement training (Rescorla, 2007). SR can be viewed as a case of proactive interference (Bouton, 1993). Following a resting period, an outdated expectancy (memory A) is retrieved and interferes with the ability of a more recently formed expectancy (memory B) to guide behavior. Accordingly, if consummatory suppression in the cSNC situation reflects the formation of an egocentric memory of frustration (memory A) that becomes counterconditioned during the postshift sessions (memory B), then the interpolation of a resting period after the recuperation of consummatory behavior should lead to the SR of the behavioral suppression typical of the first downshift session (memory A). Alternatively, if there is no egocentric memory, but only allocentric memories of 32% and 4%, then there can be no egocentric-based proactive interference and no SR of the consummatory suppression typical of the first downshift session. In such a case, proactive interference (32% expectation) would be based on the retrieval of the more remote 32% sucrose expectation, which would interfere with the retrieval of the more recently acquired memory of the 4% sucrose. Experiments 1–3 investigated the possibility that a resting period interpolated between the last postshift session and a final test session would provide evidence of the SR of cSNC, that is, of a relapse of consummatory suppression typically observed during the initial postshift sessions. Experiment 1 and 2 varied resting period (24, 96, or 336 h) interpolated following complete recuperation and incomplete recuperation. Experiment 3 utilized the opioid-receptor antagonist naloxone (2 mg/kg), known to enhance cSNC following a 96-h resting period. To further investigate SR in

consummatory situations and elucidate the results of Experiments 1–3, Experiments 4–5 looked for evidence of egocentric memory during cE, a preparation known to induce SR across sessions, using naloxone administration as a probing tool.

Experiment 1

Different groups of rats received training in two conditions, 32 → 4 vs. 4 → 4, where numbers refer to the concentration of sucrose solutions. Once recuperation from cSNC was complete, rats in each condition were randomly assigned to 24-, 96-, or 336-h resting periods. After the resting period ended, all rats received two additional sessions of access to the 4% solution. It was predicted that downshifted subjects tested after 96- and 336-h resting periods, but not after a 24-h resting period, would demonstrate SR of cSNC, operationalized as a significant consummatory suppression on testing sessions relative to the unshifted control exposed to the same resting period. It was also predicted that SR will be greater after a resting period of 336 h than after one of 96 h given that SR of fear increases with the length of the resting period (Quirk, 2002).

Method

Subjects

The subjects were 60 experimentally naïve, Long–Evans hooded male rats derived from Harlan (Indianapolis, IN), approximately 90 days old at the start of the experiment. Rats were housed under a 12:12 h light:dark cycle (lights on at 07:00 h) and were deprived of food to 81–84% of the free-food weight. Water was continuously available in each individual cage. Animals were trained during the light phase of the daily cycle. All the subjects had served as part of saline control groups for other cSNC studies and received preshift and postshift training as part of those experiments. All the rats also received a single saline injection (1 ml/kg in volume) administered before session 11.

Apparatus

Training was conducted in four conditioning boxes (MED Associates, St. Albans, VT) constructed of aluminum and Plexiglas. Each box was 29.3-cm long, 21.3-cm high, and 26.8-cm wide. The floor was made of steel rods 0.4 cm in diameter and 1.6 cm apart running perpendicular to the feeder wall. A bedding tray filled with corncob bedding was placed below the floor to collect feces and urine. Against the feeder wall was an elliptical perforation 1-cm wide, 2-cm high, and 4-cm from the floor. A sipper tube, 1 cm in diameter, was inserted through this hole. When fully inserted, the sipper tube was flush against the wall. A computer located in an adjacent room controlled the presentation and retraction of the sipper tube, and detected contact with the sipper tube by way of a circuit involving the steel rods in the floor. Each conditioning box was placed in a sound-attenuating chamber that contained a light, a speaker to deliver white noise, and a fan for ventilation. Together, the speaker and fan produced noise with an intensity of 80.1 dB (SPL, Scale C).

Procedure

Training lasted a total of 20 daily sessions. Each rat was assigned to one of the four conditioning boxes and always trained in that box. The order of training of 4-rat squads varied across days. After each session, conditioning boxes were wiped with a damp paper towel, feces removed, and bedding material replaced as needed. The light, white noise, and fan were constantly on during training sessions. The 20 sessions were divided into a preshift phase (10 sessions), a postshift phase (8 sessions), and a SR phase (2 sessions). Rats were randomly assigned to one of two groups during the preshift and postshift phases (downshifted, unshifted). After the postshift sessions and before SR testing, triplets of downshifted rats matched in their postshift performance were randomly assigned to one resting period: 24, 96, and 336 h. The unshifted controls were assigned likewise.

Thus, there were six groups in this experiment ($n = 10$). For the three 32 → 4 groups (Groups 32/336, 32/96, and 32/24), the 10 preshift sessions involved access to a 32% sucrose solution (w/w, prepared by mixing 32 g of commercial sugar for every 68 g of distilled water). The three 4 → 4 groups (Groups 4/336, 4/96, and 4/24) received access to the 4% sucrose solution during the 10 preshift ses-

sions (w/w, prepared by mixing 4 g of sugar for every 96 g of distilled water). For the eight postshift sessions and the two SR sessions, all rats received access to the 4% solution.

Each session started with a variable interval of 30 s (range: 15–45 s). At the end of this interval, the sipper tube was automatically presented. A recording period started when a rat contacted the sipper tube and lasted 5 min. Retraction of the sipper tube was followed by a variable interval of 30 s (range: 15–45 s). The dependent variable, goal-tracking time, was the cumulative amount of time (measured in 0.05-s units) in contact with the sipper tube. Previous research shows that goal-tracking time correlates significantly and positively with amount of 32% and 4% sucrose consumed across training sessions (Mustaca et al., 2002). Furthermore, this measure provides a less variable assessment of consummatory behavior, both across sessions and subjects, under the conditions used in our laboratory. Scores were subjected to conventional analysis of variance and LSD post hoc tests were used for pairwise comparisons as needed. The alpha value was kept at the 0.05 level for all statistical tests.

Results and discussion

Fig. 1 shows the overall results, in terms of goal-tracking time as a function of session. Two rats assigned to the 32 → 4 condition were eliminated for failing to show any performance decrement on session 11. There is no basis to expect SR in the absence of response suppression during the initial downshift session. Preshift performance was similar across conditions receiving access to 32% or 4% sucrose, a result not unlike that often observed in these experiments (e.g., Flaherty, 1996). A Sucrose × Session (1–10) analysis indicated a main effect of session, $F(9,504) = 145.72$, $p < .001$, and a preshift session by sucrose interaction, $F(9,504) = 2.22$, $p < .03$. Fig. 1 also shows the cSNC effect that ensued during postshift sessions. Performance matching yielded similar performance levels in both downshifted and unshifted groups. A Contrast × Session (11–18) analysis indicated a main effect of session, $F(7,392) = 31.81$, $p < .01$, a main effect of contrast, $F(1,56) = 19.41$, $p < .01$, and an interaction between contrast and postshift session, $F(7,392) = 22.98$, $p < .01$.

Further one-way analyses of variance followed by LSD pairwise comparisons were calculated to determined group effects on some key sessions, including sessions 11 (first postshift), session 18 (last postshift), and sessions 19 and 20 (SR testing). On session 11, there was a significant difference across groups, $F(5,57) = 12.95$, $p < .01$. In turn, each of the three pairs of downshifted–unshifted groups assigned to the three resting periods also differed significantly. Thus, Groups 32/24 and 4/24, 32/96 and 4/96, and 32/336 and 4/336 were all significantly different, $ps < .01$. This provides evidence of

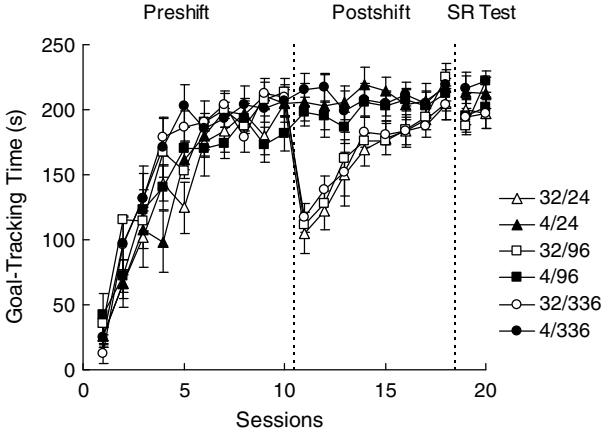

Fig. 1. Mean goal-tracking time (±*SEM*) plotted as a function of session and group from Experiment 1. In group names, 32 and 4 refer to the concentration of the sucrose solution used during preshift sessions 1–10. All animals received 4% solution during postshift and spontaneous recovery (SR) test sessions. 24, 96, and 336 refer to the length (in hours) of the resting period between sessions 18 and 19. The resting period was interpolated following complete recovery from cSNC on session 18.

cSNC effects. On session 18, a similar analysis failed to find any differences among the groups, $F < 1$, thus providing evidence consistent with complete recuperation from cSNC. The main results correspond to the two sessions of SR testing, sessions 19 and 20. As seen in Fig. 1, there was no clear evidence of SR at any value of the resting-period manipulation in either session. One-way analyses failed to show group differences in either session, Fs < 1.

These results provide no support for the hypothesis that cSNC would spontaneously recover after resting periods of 96 and 336 h, compared with a 24-h resting-period control. Relative to unshifted groups, downshifted groups exhibited both cSNC and recuperation of normal levels of goal tracking during postshift sessions, but there were no signs of SR of consummatory suppression.

Experiment 2

In his evaluation of SR, Rescorla (2004) concluded that SR is reduced following repeated extinction experience. Experiment 2 extrapolated this logic to the present situation by reducing the number of postshift sessions before interpolating the resting period. It was predicted that downshifted animals receiving 3 (rather than 8) postshift sessions and tested after a 96-h resting period would demonstrate SR, operationalized as greater consummatory suppression in Group 32/96 than in Group 4/96, but similar levels of consummatory behavior in Groups 32/24 and 4/24, during SR testing.

Method

Subjects and apparatus

The subjects were 32 experimentally naïve, Long–Evans hooded rats (16 males, 16 females), derived from Harlan (Indianapolis, IN), and approximately 90 days old at the start of the experiment. Housing, food deprivation, and training boxes were as described in Experiment 1.

Procedure

Training lasted 15 daily sessions and was similar to Experiment 1 with the following exceptions. Rats were randomly assigned to one of two conditions: 32% or 4% sucrose solution. They received 10 daily sessions of preshift training identical to those described in the previous experiment. The postshift phase was shortened from 8 to 3 sessions. Thus, all the groups received a total of 13 sessions before SR testing. Following session 13, the downshifted rats were matched in terms of their postshift performance and randomly assigned to one of the two conditions: 24- or 96-h resting period. A 96-h resting period was selected on the basis that previous investigations of SR had found significant SR following a 96-h interval (e.g., Quirk, 2002). The 336-h interval was excluded mainly on practical grounds. The unshifted controls were randomly assigned likewise. This gave rise to four groups ($n = 8$): Groups 32/96, 32/24, 4/96, and 4/24. Following their respective resting period, animals underwent SR testing on sessions 14 and 15. All other training parameters and procedures were as described in Experiment 1.

Results and discussion

Fig. 2 shows the overall results, in terms of goal-tracking time as a function of session. None of the analyses incorporating sex as a factor yielded significant main effects, so this factor was not included in the analyses reported below. There was again no apparent difference in the preshift performance of groups exposed to 32% or 4% sucrose. A Sucrose × Session (1–10) analysis indicated a main effect of preshift session, $F(9,270) = 56.28$, $p < .001$, but not sucrose or sucrose by session interaction, Fs < 1. Clear evidence of cSNC was found again during the postshift sessions. Goal tracking increased in downshifted groups during postshift sessions, but not to the level of unshifted controls, as was expected given the limited number of postshift sessions. A Contrast × Session (11–13) analysis indicated a main effect of postshift session, $F(2,60) = 4.61$, $p < .02$, a main effect of contrast, $F(1,30) = 25.04$, $p < .001$, and a significant interaction of these factors, $F(2,60) = 3.17$, $p < .05$.

A one-way analysis of variance on session 11 showed significant group differences, $F(3,31) = 6.87$, $p < .01$. LSD pairwise comparisons revealed significant differences between Groups 32/24 and 4/24,

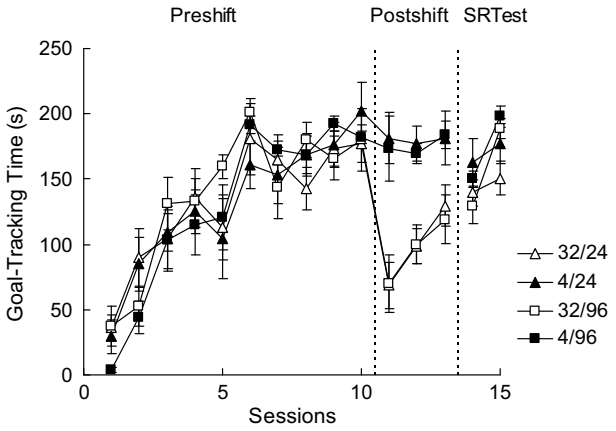

Fig. 2. Mean goal-tracking time (±*SEM*) plotted as a function of session and group from Experiment 2. The resting period was interpolated following partial recovery from cSNC. See legend of Fig. 1 for further details.

and between Groups 32/96 and 4/96, *p*s < .01. Thus, the strength of the cSNC effect was equivalent in both groups. A similar analysis calculated on the scores of session 12 revealed a similar pattern, $F(3,31) = 6.08$, $p < .01$, and similar pairwise differences between Groups 32/24 and 4/24 and Groups 32/96 and 4/96, *p*s < .05. However, an analysis of session 13 failed to indicate a significant group difference, $F(3,31) = 2.86$, $p = .055$, indicating some degree of recovery from cSNC.

Again, the main results were those of the SR test, sessions 14 and 15. As shown in Fig. 2, none of the previously downshifted groups exhibited a decrement in consummatory behavior relative to the last postshift session before the resting period. Rather, their performance seemed to follow the recuperative trend of the postshift sessions. A one-way analysis on goal-tracking times for sessions 14 and 15 failed to show significant group differences on session 14, $F < 1$, or 15, $F(3,31) = 2.05$, $p > .05$. These results fail to provide any evidence of the SR of cSNC following incomplete recuperation of consummatory behavior after the incentive downshift.

Experiment 3

Experiments 1 and 2 failed to provide evidence of the SR of cSNC either after complete or after incomplete recuperation of normal levels of consummatory behavior during the postshift phase. Experiment 3 is based on the assumption that the lack of evidence for SR of cSNC is caused by a weakly active egocentric memory of the downshift experience at the time of testing. Perhaps the SR of cSNC could be induced by a pharmacological manipulation. As mentioned in the introduction, the nonselective opioid-receptor antagonist naloxone administered during the downshift sessions enhanced cSNC. One interpretation of this effect (Pellegrini et al., 2005) suggests that blockage of the opioid system by naloxone intensifies the aversive hedonic value of the downshift experience, thus prolonging the recuperative process during postshift sessions. Thus, the subthreshold reactivation of the egocentric memory during SR testing might become detectable in terms of consummatory behavior by the enhancing effect of naloxone on the aversive hedonic content of that memory. Naloxone should have no suppressive effect during the SR test in unshifted controls never exposed to the downshift event.

Method

Subjects and apparatus

The subjects were 34 experimentally naïve, Long–Evans hooded male rats derived from Harlan (Indianapolis, IN), and approximately 90 days old at the start of the experiment. Some rats ($n = 12$) received the current preshift and postshift training as part of saline control groups in another cSNC

experiment. In that experiment, a single saline injection (1 ml/kg in volume) was administered following session 11. The rest ($n = 22$) did not receive saline administration. Housing, food deprivation, and training boxes were as described in Experiment 1.

Procedure

Behavioral training lasted for a total of 20 daily sessions and was identical to that described in Experiment 1, with the following exceptions. Rats were randomly assigned to training with either 32% or 4% sucrose during preshift sessions. Three rats in each group had received a saline injection after trial 11. After the last postshift session, but before SR testing, downshifted and unshifted rats were matched in terms of their postshift performance and randomly assigned to one of the following conditions. Groups 32/Nlx ($n = 8$) and 4/Nlx ($n = 9$) received an injection of naloxone (2 mg/kg, ip). Groups 32/Sal ($n = 8$) and 4/Sal ($n = 9$) received an injection of saline (equal volume, ip). The 2 mg/kg dose of naloxone was chosen on the basis of prior research in the same behavioral preparation (Pellegrini et al., 2005). All rats received the appropriate injection 15 min prior to SR testing on session 19. No drugs were administered before session 20. Drugs were purchased from Sigma–Aldrich Chemicals (Saint Louis, MO). All groups were exposed to the 96-h resting period. In addition to recording the goal-tracking time (0.05-s units) as described previously, within-session goal-tracking times were recorded in 5-s bins to provide information on the possible occurrence of SR across postshift sessions.

Results and discussion

Fig. 3 shows the overall results in terms of goal-tracking time as a function of sessions. As in previous experiments, no clear differences were found in the preshift performance of these groups as a function of sucrose concentration. A Sucrose × Session (1–10) analysis indicated a main effect of session, $F(9,288) = 69.07$, $p < .01$, but no effect of sucrose or of the sucrose by session interaction, Fs < 1. A clear cSNC effect emerged during the postshift phase, followed by the recuperation of consummatory performance. A Contrast × Session (11–18) analysis showed a main effect of session, $F(7,224) = 13.07$, $p < .01$, a main effect of contrast, $F(1) = 6.87$, $p < .02$, and of contrast by session interaction, $F(7,224) = 4.71$, $p < .001$.

A one-way analysis of variance on session 11 indicated a significant group effect, $F(3,33) = 6.19$, $p < .01$. LSD pairwise comparisons revealed significant differences between Groups 32/Nlx and 4/Nlx, and between Groups 32/Sal and 4/Sal, ps < .01. These results provide evidence of cSNC effects in both pairs of groups. A second one-way analysis calculated on session 18 indicated nonsignificant group differences, $F < 1$. Thus, consummatory performance had recuperated by the end of the postshift phase.

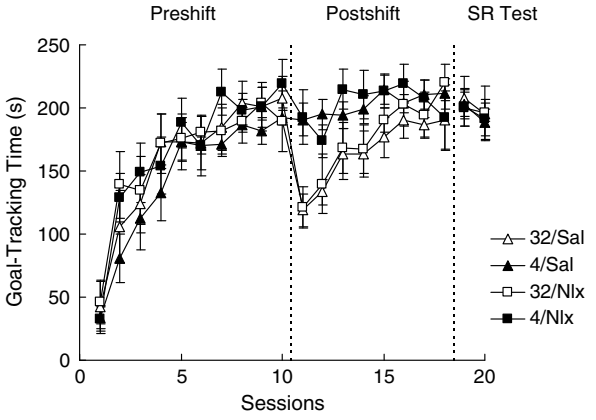

Fig. 3. Mean goal-tracking time (±*SEM*) plotted as a function of session and group from Experiment 3. Nlx and Sal refer to the drug administered 15 min before session 19 of spontaneous recovery (SR) testing, either naloxone (2 mg/kg, ip) or an equal-volume saline injection (Sal, ip). See legend of Fig. 1 for further details.

The main results concern the performance on SR testing on sessions 19 and 20. As shown in Fig. 3, there was no evidence that the administration of naloxone on session 19 had induced any detectable amount of consummatory suppression. A one-way analysis calculated on the data from session 19 failed to show significant group differences, $F < 1$. Similarly, an analysis of session 20 failed to reveal any group effect, $F < 1$.

Whereas the results of Experiment 3 (as well as those of Experiments 1 and 2) provided no evidence of SR of cSNC, a within-session analysis of the performance of these rats on postshift and SR testing sessions revealed the consistent recovery of consummatory performance across sessions. Fig. 4 shows the within-session scores in 100-s bins across all postshift sessions and session 19 after naloxone administration. Clearly, downshifted rats started each postshift session exhibiting a level of consummatory performance that looked like that of unshifted controls and only subsequently showed evidence of response suppression. (Similar within-session performance was observed in Experiments 1 and 2.) A Contrast × Drug × Bin × Session analysis for postshift sessions 11–18 revealed several interesting effects. There were within-subject main effects of session, $F(7,210) = 14.30$, $p < .01$, and bin, $F(2,60) = 27.05$, $p < .01$, and a between subject effect of contrast, $F(1,30) = 6.61$, $p < .02$. There were the following within-subject, two-way interactions: session by contrast, $F(7,210) = 4.49$, $p < .001$; bin by contrast, $F(2,60) = 34.57$, $p < .001$; and session by bin, $F(14,420) = 3.21$, $p < .001$. There was also the triple interaction (session by bin by contrast), $F(14,420) = 4.32$, $p < .01$. A similar Contrast × Drug × Bin analysis for postshift session 19 was conducted. There was a within-subject effect of bin, $F(2,60) = 13.14$, $p < .001$. In none of these analyses there was a drug effect.

The results of Experiments 1–3 failed to provide support for the hypothesis that cSNC is subject to relapse after a postrecuperative period of rest. Such a relapse would have been analogous to the familiar phenomenon of SR readily obtained in fear conditioning and other preparations (e.g., Quirk, 2002). In fact, these results provide support for an alternative view based on the hypothesis that the cSNC situation requires only allocentric memory (of the original 32% sucrose and the newer 4% sucrose events) combined with primary frustration to account for consummatory suppression during the early postshift trials. Thus, SR involves the reactivation of the preshift memory of the 32% sucrose, which when compared to the current 4% sucrose induces primary frustration and thus consummatory suppression. The conflict may then be described more as an approach-rejection conflict than as an approach-avoidance conflict (see General discussion for an elaboration of the distinction between "rejection" and "avoidance"). Whereas these failures must be taken with caution, the range of resting periods (24–336 h), the number of postshift sessions (3–8), and the attempt to induce SR via naloxone administration suggest that the type of consummatory suppression induced by incentive downshift is unlikely to recover some of its strength after a resting period. The within-session data and prior

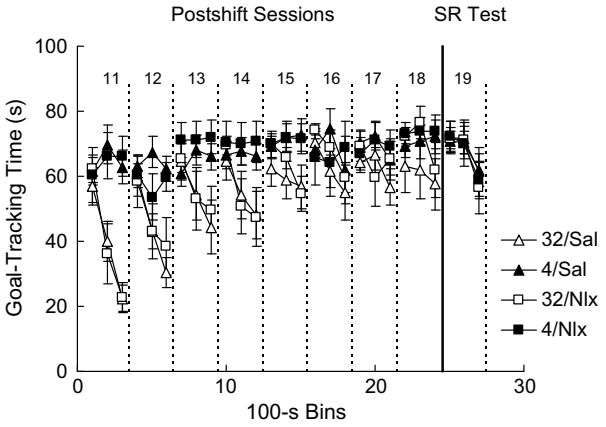

Fig. 4. Within-session performance for all postshift sessions (11–19) from Experiment 3. Mean goal-tracking time (±*SEM*) was plotted as a function of 100-s bins. Nlx and Sal refer to the drug administered 15 min before session 19 of spontaneous recovery (SR) testing, either naloxone (2 mg/kg, ip) or an equal-volume saline injection (Sal, ip). See legend of Fig. 1 for further details.

research (Mustaca et al., 2002) suggest that such failures of SR are not due to an intrinsic failure of consummatory behavior to exhibit SR. Rather, to the extent that the SR of consummatory behavior reveals high levels of goal tracking early in the extinction sessions, it suggests that while the expectancy of the preshift incentive recovers from session to session, the expectancy of the incentive downshift (the session 11 experience) is not subject to relapse.

Experiment 4

Experiments 1–3 failed to show SR of cSNC following complete recovery, partial recovery, and administration of naloxone. However, this does not mean that SR is absent from consummatory preparations using sucrose solutions, as shown in Fig. 4. Mustaca et al. (2002) reported a similar type of SR effect across sessions in a cE situation. In this instance, SR is operationally defined as the increase in the strength of consummatory behavior early in one session (after a 24-h resting period) relative to the level exhibited late in the previous session (without a resting period). In that experiment, extinction performance was analyzed in 1-min bins within each extinction session. In such a design, and assuming no decrement in the level of SR across sessions (a result to be expected with relatively few extinction sessions, as in the current Experiments 4–5), SR is statistically confirmed if there is a significant decrease within the session, combined with a nonsignificant session by bin interaction (i.e., similar within-session decrease across sessions).

The present experiment was designed to determine whether this type of SR, known to occur from a previous report (Mustaca et al., 2002), was susceptible to the effects of naloxone. The rationale is the same used in the introduction to Experiment 3. Thus, if incentive downshift leading to consummatory suppression during the first extinction session triggers the acquisition of an egocentric memory, it is expected that naloxone would enhance any residual aversive component of that memory and therefore lead to a reduction of SR (i.e., low levels of consummatory behavior early in the extinction session). In fear conditioning, naloxone attenuates the extinction of fear conditioning, suggesting that opioid blockage enhances the aversive hedonic properties of fear (McNally & Westbrook, 2003). Interestingly, if incentive downshift does not trigger memory formation, but it contains an aversive component that recruits during the session (e.g., primary frustration; Amsel, 1992), then naloxone is expected to increase its aversiveness too. But in such a case, naloxone would reduce consummatory behavior later in the extinction session, without affecting SR. In such a case, SR of consummatory behavior would be attributable to the reactivation of the preextinction memory of the incentive.

Method

Subjects and apparatus

The subjects were 34 Long–Evans hooded male rats derived from Harlan (Indianapolis, IN), approximately 120 days old at the start of the experiment. These rats were those used in Experiment 3. Housing, food deprivation, and training boxes were as described in Experiment 1.

Procedure

Immediately after session 20 of Experiment 3, all groups continued their exposure to the 4% solution for an additional three sessions. Rats were reassigned to two groups matched with respect to prior contrast experience and drug treatment (recall that naloxone had no effect on consummatory behavior in Experiment 3). Thus, two groups were established ($n = 17$): Groups Sal and Nlx. During the following three sessions, rats in both groups were exposed to an empty sipper tube in otherwise identical training sessions. Naloxone (2 mg/kg, ip) and saline (equal volume) were administered 15 min before the second and third extinction sessions (i.e., no SR can be assessed on the first extinction session). Other aspects of training were identical to those described in Experiment 1.

Results and discussion

One rat from Group Sal died during the course of this experiment and its data were removed from all analyses. Within-session data were integrated in three 100-s bins for each rat. Because of the com-

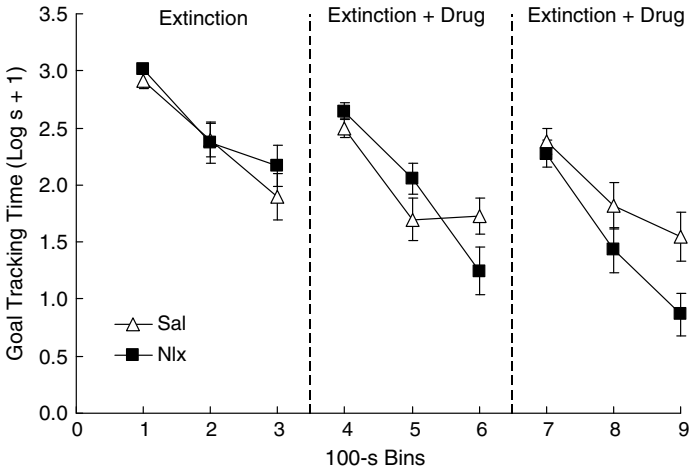

Fig. 5. Mean goal-tracking time ($\log_{10}$ plus 1; ±*SEM*) plotted as a function of 100-s bins from Experiment 4. Following Experiment 3, all subjects received access to 4% sucrose for three additional daily sessions and were then assigned to the two groups so as to match previous drug exposure. During extinction sessions, subjects were presented with an empty sipper tube. Either naloxone (Nlx, 2 mg/kg, ip) or an equal-volume saline solution (Sal, ip) were administered 15 min before the second and third extinction sessions (no drugs were administered before the first extinction session).

mon occurrence of low goal-tracking times in the cE preparation and to improve normality, individual scores (plus a constant of 1 to avoid zeros) were transformed to the $\log_{10}$. Fig. 5 shows the extinction performance of both groups for each session in 100-s bins. Goal-tracking times decreased at a similar rate across groups during the first session and while animals were untreated, confirming that groups were matched in terms of initial extinction performance. A Drug × Bin analysis of the first extinction session indicated a main effect of bin, $F(2,62) = 53.53$, $p < .001$, but no effect of naloxone, $F < 1$, or of the drug by bin interaction, $F(2,62) = 1.27$, $p > .28$.

Consummatory behavior increased in the first bin of the second session to decrease again in the rest of that session; this cycle was repeated in the third session. Naloxone administration during the second and third extinction sessions did not affect the initial level of performance in each session (i.e., no effect on SR of consummatory behavior). However, naloxone administration reduced the level of responding in the third bin of both sessions, relative to the saline group. A Drug × Session × Bin analysis of the second and third sessions revealed the following results. SR was confirmed by a significant decrease in responding across bins (i.e., within-session extinction), $F(2,62) = 55.93$, $p < .001$, combined with a nonsignificant session by bin interaction, $F < 1$. Thus, the within-session decrease in consummatory performance was repeated across the two sessions. There was also a significant decrease in responding across sessions, $F(1,31) = 6.46$, $p < .02$, and a significant bin by drug interaction, $F(2,62) = 5.06$, $p < .01$. One-way ANOVAs for each bin were calculated to determine the source of the bin by drug interaction; these analyses revealed a significantly lower response score in the naloxone group, compared to the saline control, on bin 9, $F(1,31) = 5.71$, $p < .03$. The drug effect was nonsignificant for other bins, Fs$(1,31) < 3.21$, ps $> .08$.

Experiment 5

Experiment 4 extended the findings reported by Mustaca et al. (2002) by demonstrating that the opioid-receptor antagonist naloxone facilitates extinction of consummatory behavior without interfering with the SR of such behavior across sessions. This facilitation of extinction occurred at the end of the extinction session, rather than the earlier portions, suggesting that early responding may be under the control of the allocentric memory of the preextinction incentive. One limitation of these findings is the use of sessions of continuous access to the sipper tube that forced a view of SR based on

a relatively arbitrary breaking of bins. In typical conditioning experiments, training is administered in discrete trials that have a clear onset and offset. Experiment 5 was designed to extend the findings of the previous experiment to a discrete-trial consummatory procedure. The total 300 s of access to the sipper tube were broken into six 50-s trials separated by an average intertrial interval of 50 s. Trials were generated by the automatic presentation and withdrawal of the sipper tube.

Method

Subjects and apparatus

The subjects were 16 Long–Evans hooded rats (8 males, 8 females) derived from Harlan (Indianapolis, IN), approximately 120 days old at the start of the experiment. These animals had been originally assigned to the 24-h resting period in Experiment 2 and received training for the present experiment immediately after the termination of that experiment. Housing, food deprivation, and training boxes were as described in Experiment 1.

Procedure

After the conclusion of Experiment 2, each rat in the 24-h resting-period condition (both downshifted and unshifted animals) was assigned to one of two groups, Groups Nlx and Sal ($n = 8$), in such a way that prior experience was matched across groups. Behavioral training lasted for a total of nine daily sessions. Each session included six trials, each lasting 50 s and separated by an intertrial interval of 50 s on average (range: 25–85 s). Rats received six daily sessions of access to the 4% sucrose solution followed by three daily extinction sessions of access to an empty sipper tube. The total time the sipper tube was available per session was equal to that experienced during the typical training sessions of Experiment 2 (i.e., 5-min daily sessions). Naloxone and saline were administered as described in Experiment 4. Other aspects of the procedure were as described in Experiment 1.

Results and discussion

Fig. 6 shows the data for each group in 2-trial blocks (equivalent to the 100-s bins used to analyze the results of Experiment 4). Extinction was similar across groups during the first session, when no treatment was administered. None of the analyses incorporating sex as a factor yielded significant ef-

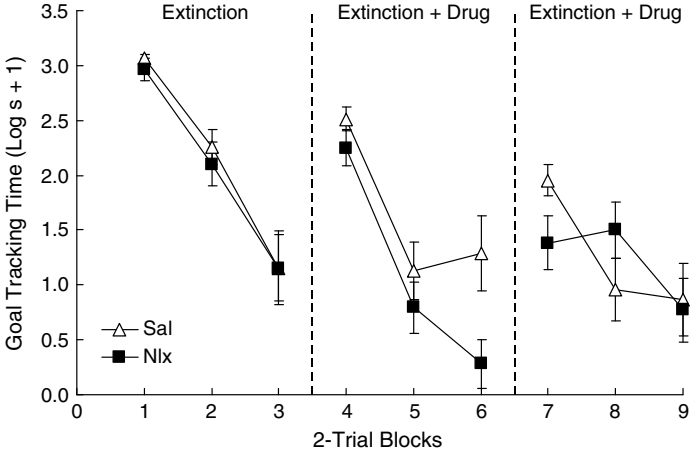

Fig. 6. Mean goal-tracking time ($\log_{10}$ plus 1; ±*SEM*) plotted as a function of 2-trial blocks from Experiment 5. Each 2-trial block involves 100 s of sipper-tube presentation. Unlike in Experiment 4, the sipper tube was presented in discrete trials, each 50-s long in duration. Extinction sessions were preceded by six sessions of access to 4% sucrose. See legend of Fig. 5 for further details.

fects for either the main effect or any interaction involving sex. Therefore, sex was eliminated from the main analyses. A Drug × Block analysis of the first extinction session indicated a main effect of block, $F(2,28) = 49.43$, $p < .001$, but no effect of drug or the drug by block interaction, Fs < 1. Naloxone also increased the rate of cE, but only during the second extinction session. A Drug × Session (2–3) × Block analysis revealed a main effect of block, $F(2,28) = 25.92$, $p < .001$. There was a significant interaction between session and block, $F(2,28) = 8.44$, $p < .01$. There was also a triple interaction between session, block, and drug, $F(2,28) = 3.92$, $p < .04$. The main effect of drug was nonsignificant, $F(1,14) = 1.81$, $p > .20$. One-way analyses of each 2-trial block revealed that the source of the triple interaction was a significantly lower extinction performance in the naloxone group relative to the saline control on block 6, $F(1,15) = 6.20$, $p < .03$. These results suggest that naloxone facilitated extinction, but had no effect on SR.

General discussion

Five experiments investigated SR in two consummatory preparations involving surprising nonreward: cSNC and cE. Experiments 1–3 evaluated the possibility that the cSNC effect would relapse after a resting period without access to the training situation following the recuperation of consummatory levels similar to those of unshifted controls. Experiment 1 revealed no evidence of postrecuperatory SR of cSNC after resting periods of 24, 96, or 336 h. Experiment 2 failed to find evidence of SR of cSNC after partial recuperation of control levels of consummatory behavior during postshift sessions. Experiment 3 uncovered no evidence of postrecuperatory SR of cSNC in animals treated with the nonselective opioid antagonist naloxone—a drug known to enhance cSNC (Pellegrini et al., 2005). Whereas these results cast doubt on the possibility that the cSNC phenomenon is subject to relapse, this failure cannot be attributed to an inability of consummatory behavior to demonstrate SR. Indeed, consummatory behavior was known to exhibit a form of SR from cE experiments involving transitions from access to sucrose solution to access to an empty sipper tube (Mustaca et al., 2002). In addition, a within-session analysis of Experiment 3 data yielded evidence of SR across postshift sessions (Fig. 4). During postshift sessions, cSNC occurs in downshifted rats because their behavior recovers levels analogous to those of preshift sessions, when they received 32% sucrose, to decrease toward the end of the session. This decrease is gradually reduced across postshift sessions as consummatory behavior recuperates a level similar to that of unshifted controls. It is tempting to argue that cSNC occurs because the initial portions of each postshift session reactivate a memory of the 32% solution received during preshift sessions, thus setting the conditions for a surprising reduction in incentive value. A test of this assertion would require conditions that generate different preshift performance levels, so as to determine whether the level of performance during early portions of postshift sessions is actually higher than that exhibited by unshifted controls. Unfortunately, the current study could not differentiate performance associated with expectancies of 32% and 4% sucrose because the initial levels of responding were statistically indistinguishable, even when within-session data were analyzed in 5-s bins—the highest temporal resolution available. Therefore an interpretation of the cSNC phenomenon based on the SR of preshift sucrose expectations must be considered preliminary.

Experiments 4 and 5 evaluated the SR of consummatory behavior across sessions of extinction separated by 24-h intervals. As already mentioned, a previous set of experiments provided the initial evidence of 24-h SR during cE (Mustaca et al., 2002). The present focus was on the effects of the nonselective opioid-receptor antagonist naloxone. Several experimental results in the fear conditioning literature suggest that naloxone administration increases the intensity of the state of fear induced by a conditioned stimulus paired with peripheral pain. For example, naloxone (2.5 mg/kg, sc) administered before training sessions retarded the decrease of freezing responses normally observed during fear extinction (McNally & Westbrook, 2003). Thus, a similar effect of naloxone is predicted for behaviors controlled by secondary frustration (see Papini, 2003). This rationale was used to test for the SR of cSNC in Experiment 3 without success and it was implemented again in Experiments 4 and 5 to test for SR during cE. If naloxone intensifies secondary frustration, then its administration should reduce SR, suppressing consummatory behavior during the early sections of the extinction sessions when the egocentric memory of the downshift is reactivated. However, if naloxone intensifies primary frus-

tration, then naloxone should suppress consummatory behavior in later portions of the extinction sessions. Whether extinction was carried out in a single 5-min session (Experiment 4) or in discrete trials (Experiment 5), naloxone administered before the second and third extinction sessions had no effect on SR, but it reduced the later portions or trials of extinction sessions. Naloxone was not administered before the first extinction session because no SR can be measured in this session. In addition, the absence of group differences in the first extinction session suggests that the effects of naloxone are not attributable to biased assignment of rats to the two groups. Furthermore, because naloxone accelerated extinction in both procedures, the lack of an effect on SR cannot be attributed to an ineffective dose. These results suggest that the psychological mechanism underlying the effect of naloxone on cE is related to the enhancement of primary frustration, rather than to the development of an egocentric memory of the downshift (i.e., secondary frustration).

As pointed out in the introduction, incentive downshift in the cSNC situation has been previously assumed to involve both an unconditioned emotional response of primary frustration driving rejection of the downshifted solution and a conditioned emotional response of secondary frustration that contributes the avoidance component of the approach-avoidance conflict presumed to develop during the second postshift trial (Papini, 2003; Wood et al., 2005). This theoretical analysis is consistent with the differential effects of both benzodiazepine anxiolytics and opioids when administered before the first vs. second postshift sessions (see Flaherty, 1996; Papini, 2006). However, as pointed out in the introduction, these drug effects do not unambiguously demonstrate the presence of aversive conditioning resulting from the downshift experience in the cSNC situation. One implication of the absence of SR in the cSNC situation is that there might not be any memory to restore. SR involves the restoration of a previously acquired response that has undergone extinction or counterconditioning (Bouton & Peck, 1992; Quirk, 2002). Thus, the assumption that an aversive egocentric memory formed during the initial downshift experience (session 11) and subsequently counterconditioned during postshift sessions is called into question by the present results. Consistent with this interpretation, the latency to the first contact with the sipper tube was reported to be unchanged during postshift sessions in a cSNC experiment, indicating that the egocentric memory, if any, is not elicited by contextual cues and does not interfere with initial approach behavior (Flaherty, Hrabinski, & Grigson, 1990). In addition, if rats acquire an egocentric memory during the initial downshift event, anticipation of frustration should enhance suppression early in the second postshift session. The evidence from Experiment 3 indicates that drinking behavior is suppressed progressively through the second postshift session, while little suppression occurs at the start of the session. This suggests that the egocentric memory may not be present, or if it is, its influence on consummatory behavior cannot be detected with the current procedures.

An additional possibility is that the frustration memory is present, but is overwhelmed by the preshift memory for 32% sucrose (i.e., proactive interference). Compared to the 10 preshift trials with 32%, there are fewer trials that invoke primary frustration—mainly trial 11. Furthermore, an account of cSNC based on Amsel's (1992) frustration theory includes conflict between the approach tendency to the sucrose and the avoidance of frustration. Miller (1944) described conflict in terms of gradients that become stronger as the goal becomes closer, and characterized avoidance gradients as steeper than approach gradients. When the goal is further away, approach gradients are stronger, and thus control behavior. As postshift trials progress temporally further from each of the conflicting memories in cSNC, it is possible that the memory for either the 32% or the 4% sucrose (which determines the approach gradient) is stronger than the egocentric memory for frustration (which determines the avoidance gradient). Thus, the SR of approach consummatory behavior conceals the SR of avoidance based on secondary frustration. Available evidence suggests that when rats are trained in a conflict situation with temporal gradients, they do not exhibit spontaneous recovery of either memory following extinction (Rigby, 1954). Overall, because the establishment of a strong memory of 32% sucrose is necessary to elicit primary frustration, the interference between the two memories during cSNC might be a fundamental impediment to determine the presence of SR of cSNC.

To determine whether a pattern of results similar to that observed for naloxone in cE (Experiments 4–5) also applies to the effects of naloxone on cSNC, a within-session analysis was carried out on the results of a previous experiment reported in Pellegrini et al. (2005, Experiment 1). These within-session data were not originally reported and remained in storage until the results of the present Exper-

iments 4–5 became available. In the original experiment, groups of rats received access to either 32% sucrose or 6% sucrose. Thus, the downshift manipulation involved a change from 32% to 6% sucrose. These parameters were chosen to minimize floor effects on the expectation that naloxone would cause further suppression of consummatory behavior after the downshift. Naloxone (2 mg/kg, ip) was administered 15 min before the first and second postshift sessions (see Pellegrini et al., 2005, for additional procedural information). Fig. 7 shows the within-session performance of the naloxone and saline groups (both downshifted and unshifted controls) during postshift sessions 11–14, in 5-s bins. Session 11 showed nearly normal, but still lower initial performance in the downshifted rats treated with naloxone, followed by a sharp suppression after about 10 s of exposure to the 6% solution. A transient reduction in consummatory behavior during the first half of session 11 was also evident in the unshifted control treated with naloxone. SR of preshift performance levels was observed on sessions 12 and 13 in the downshifted saline group. Although naloxone had no visible effect on performance during sessions 12 and 13 in the unshifted group, it had a clear suppressive effect in the downshifted group. Interestingly, this suppressive effect was seen already from the first 5-s bin, at the start of the session. Thus, naloxone did impair SR during the postshift sessions of a cSNC experiment. These results were confirmed statistically. A Drug (naloxone, saline) × Contrast (32%, 6%) × Session (11–14) × 5-s Bin (1–59) analysis indicated significant triple interactions for drug, contrast, and session, $F(3,81) = 4.78$, $p < .005$, and contrast, session, and bin, $F(174,4698) = 1.89$, $p < .001$. There were also significant double interactions for drug and contrast, $F(1,27) = 11.73$, $p < .003$, drug and sessions, $F(3,81) = 5.17$, $p < .01$, contrast and sessions, $F(3,81) = 25.33$, $p < .001$, contrast and bin, $F(58,1566) = 6.20$, $p < .001$, and session and bin, $F(174,4698) = 2.21$, $p < .001$. All main effects for the four factors also reached significance, Fs > 5.76, ps < .001. Other effects were not significant.

Pairwise analyses were computed for the first 5-s bin in each postshift session to determine whether naloxone affected consummatory behavior in the earliest portion of the session. A pairwise comparison shows that Group 32/Nlx was significantly suppressed on the first 5-s bin relative to Group 32/Sal only on trial 12, $F(1,13) = 27.06$, $p < .001$. The difference between these groups was not significant on trials 11, 13, and 14, Fs$(1,13) < 3.53$, ps > .08. Groups 6/Nlx and 6/Sal were not different on the first 5-s bin in any of the four postshift trials, Fs < 1. These results are consistent with the

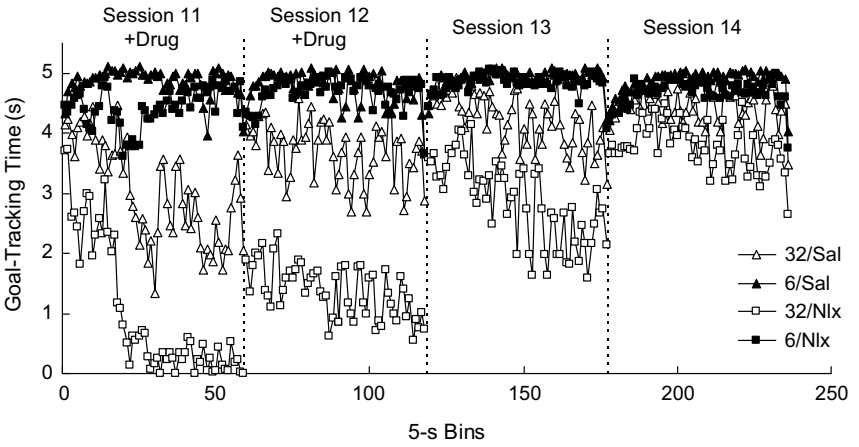

Fig. 7. Within-session performance of rats treated with naloxone (Nlx; 2 mg/kg, ip) or an equal-volume saline solution (Sal; ip) 15 min before trials 11 and 12. The original experiment was reported in Pellegrini et al. (2005)(Experiment 1). The present analysis was not included in the original publication. Downshifted groups received access to 32% sucrose for 10 preshift sessions (not shown in this figure), followed by access to 6% sucrose for an additional five sessions (four sessions are shown in this figure). Unshifted groups received access to 6% sucrose throughout the experiment. The lower concentration was 6% sucrose, rather than the usual 4% sucrose, to minimize potential floor effects derived from the predicted suppressive effects of naloxone on consummatory behavior.

presence of a short-lived egocentric memory of the downshift whose aversive value is enhanced by naloxone on trial 12.

An interpretation of the effects of naloxone on cSNC as enhancing the aversive value of an egocentric memory does not apply to the cE data of Experiments 4 and 5. If extinction led to the development of an egocentric memory mediated by the opioid system, then naloxone treatment should have reduced SR. Instead, the suppressive effect of naloxone on later portions of the extinction sessions suggests a facilitatory effect on the recruitment of unconditioned primary frustration. A possible explanation of the discrepancy between the effects of naloxone on cSNC and cE relies on the extent of the downshift. In the cSNC experiment reviewed before, the downshift was from 32% to 6% sucrose, whereas in the cE experiments reported here the downshift was from 4% sucrose to an empty tube. In iSNC, which as argued in the introduction assesses primarily the aversive egocentric memory of the downshift experience, the size of the effect is a direct function of the size of the disparity between preshift and postshift incentive magnitudes (Dilollo & Beez, 1966). In cSNC, the strength of consummatory suppression following incentive downshifts is also a direct function of the disparity between the preshift and postshift incentives (Papini & Pellegrini, 2006). Thus, it seems plausible that naloxone would have an effect on SR (i.e., on earlier portions of the extinction session) when rats are downshifted from a higher preshift sucrose concentration (e.g., 32% sucrose) to extinction.

The present failure of resting periods to induce a relapse of cSNC is analogous to a failure to find SR in passive avoidance learning reported by McAllister and McAllister (2006). Fear acquisition was measured in terms of an increased latency to cross into the initially preferred dark half of a shuttle box, previously paired with shock administration. After extinction, a resting period failed to restore the long latencies typical of acquisition performance. The passive avoidance preparation parallels cSNC in at least three respects: they are both aversive, both involve an active approach component, and they both lead to behavioral inhibition of approach behavior. Approach occurs to a dark compartment in the case of passive avoidance and to a sipper tube delivering sucrose solution in the case of cSNC. Regular fear conditioning is also aversive and requires response inhibition (i.e., freezing), and yet readily yields evidence of SR after extinction or counterconditioning (Bouton & Peck, 1992; Quirk, 2002). Similarly, situations that involve approach and avoidance responses just like cSNC and passive avoidance, also readily exhibit SR, including conditioned suppression of an appetitive baseline (Burdick & James, 1970; Estes & Skinner, 1941) and conditioned taste aversion (Mickley et al., 2007). Unlike in conditioned suppression, however, both cSNC and passive avoidance require an approach response to the site where the aversive component of the task (i.e., downshift and shock) is experienced. But so does conditioned taste aversion, which, as pointed out above, readily shows SR. Therefore, the factors responsible for the absence of SR in the cSNC situation remain undetermined.

In conclusion, the present experiments provide clear evidence of the SR of consummatory behavior during recuperation of normal levels of performance after an incentive downshift (Experiment 3) and during the extinction of consummatory behavior (Experiments 4–5). However, no evidence was found that the consummatory suppression typical of early downshift sessions in the cSNC situation recovers its strength after a resting period (Experiments 1–3). Opioid antagonism yielded evidence of modulation of primary frustration during cE (Experiments 4–5), but no evidence of inducing SR of cSNC after a resting period (Experiment 3). The only (tentative) evidence of an egocentric memory of the downshift experience was provided by a reanalysis of the postshift performance of rats treated with naloxone. These animals did exhibit a reduction of SR in the earliest portions of the second and third postshift sessions, thus suggesting that naloxone enhanced the expression of the aversive egocentric memory of the downshift. This possibility merits further examination.

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